A critical review on neonatal hyperbilirubinemia—an ayurvedic perspective

Shubhangi Rathore*, Chethan Kumar VK, Sharashchandra R

Department of Kaumarabhritya, Shri Dharmasthala Manjunatheshwara College of Ayurveda and Hospital, Udupi, Karnataka, India

Abstract

Neonatal hyperbilirubinemia is the elevation of the bilirubin level in the newborns blood, which results in yellowish staining of the skin and sclera of the newborn eyes by pigment of bile. It is due to the breakdown of RBC’s (which release bilirubin into the blood) and the immaturity of newborns liver (which cannot effectively metabolize the bilirubin and prepare it for excretion into the urine). Increased bilirubin production, reduced hepatic clearance and enhanced enterohepatic circulation are the sole causes of increased prevalence of jaundice in newborn. The science of Ayurveda is supposed to add a step in order to understand the pathophysiology of neonatal jaundice that have resemblance with clinical entity of kamala (jaundice) mentioned in Kashyapa Samhita. The concept of neonatal hyperbilirubinemia in Ayurveda can be understood in the context of Pittaja stanya dushti along with the physiological variations in the newborns leading to the raised level of unconjugated bilirubin. Therefore, the patho-physiology should be known by a pediatrician in Ayurveda based on the involvement of dosha, dhatu, mala and srotas. Hence, an attempt is made in this review to discuss about the hidden concept of pathology of neonatal jaundice described in Ayurveda. These findings to understand the concept of neonatal jaundice in Ayurveda add up to the Ayurvedic science that has been developed through ages.

1. Introduction

Newborn have unique health issues and problem due to structural and functional immaturity of various body organs depending upon their gestational age and birth weight [1]. Jaundice is the most common abnormal finding in neonates. About 60% of term and 80% of preterm babies develop jaundice in the first week of life. Untreated severe hyperbilirubinemia often signifies a serious illness [2]. Unconjugated bilirubin can cross the blood brain barrier due to many factors. These include alterations in the bilirubin binding capacity of albumin and other proteins and disruption of the blood brain barrier due to underlying conditions like asphyxia, acidosis etc. It is due to physiological polycythemia, shorter lifespan of RBC (90 days vs 120 days in adults), limited hepatic uptake, conjugation and excretion of bilirubin due to transient deficiency of receptor proteins and UDGPT enzymes in newborn especially in premature.

It is also due to paucity of bacterial flora in the gut and over activity of beta glucuronidase enzyme in the newborn. In India, physiological jaundice, immaturity, blood group incompatibility, antenatal and postnatal infections, G-6PD deficiency and breast milk jaundice are the causes of order of incidence of neonatal jaundice. Higher prevalence of jaundice is due to increased bilirubin production, reduced hepatic clearance and enhanced enterohepatic circulation in newborn. Increased bilirubin production can overwhelm the normal buffering capacity of the blood and result in the production of bilirubin acid, which is highly neurotoxic. It may cause transient encephalopathy and kernicterus which may progress over 24 h to 7 days [3].

The broad aim of this article is to provide a general outline on the description of neonatal hyperbilirubinemia from Ayurvedic perspective. This article reviews the available literature to understand the pathological changes and its manifestations in relation to neonatal hyperbilirubinemia through Ayurveda. It is written with an intention to create awareness and implement the principles mentioned in the ancient texts, suggesting an integrated approach in its management and treatment.
2. Analysis of neonatal hyperbilirubinemia in Ayurveda

By looking on to the signs and symptoms of the neonatal jaundice, it can be considered similar as that of the features of Kamala explained by Kashyapa Acharya in Kashyapa samhita. The Signs and symptoms of Kamala related to shishu (infant) are described in Vedana Adhyaya, Sutrasthan, Kashyapa Samhita are considered to be an exclusive texts on pediatrics in Ayurveda. Vedana Adhaya is written by Vrudhha Jeevaka after he prayed to Lord Kashyapa to explain the features an infant will show, when he is not able to express his pain, for the diagnosis of various diseases[4] (How a physician should know, only on the basis of clinical features, about various pains children’s who cannot narrate the symptoms.)

The symptomatology of Kamala is quite similar with jaundice of neonates, as this grantha (book) is uniquely described for Pediatrics. Kashyapacharya also described the disease as one of the lakshana’s of Revati graha [10]. The Revati graham has synonyms like Shasthi, Mukhmandika. Acharya advised to worship shasti maaata on 6th day after birth of child (neonatal period). This make clear that it can affect the neonate by producing symptoms like Neonatal jaundice.

Virechana (purigation) has been considered as the best treatment according to Kashyapa in Pittaja Stany sthiti. For “Daugandhya Dasoha” (a type of Pitta vitiated breast milk) use of Vishanika, Ajashtingi, Triphala, Rajani, Vacha with cold water is indicated.

There are two references in classical texts of Ayurveda where the jaundice in newborn can be considered-

a) Pishachi Jataharini[10]: In which the newborn dies immediately after birth and is a mansahari yapya jataharini. The word Pishachi in shabdakosha has been given the meaning as a demon which has characteristic yellow color that kills the baby on first day itself can be considered as the pathologic neonatal hyperbilirubinemia.

b) Pittika stanya dushti janya kamala: M. nidana has explained that if a baby takes pitta dusha stanya (vitiated milk), he exhibits the features like excessive sweating, loose stools, kamala roga (jaundice), thirst and increased body temperature. Charaka Acharya explains daugandyha dushti (fetid smell) and vivarnata (discoloration) as the features of pittaja ksheera dushti (Pitta vitiated breast milk) [11].

2.2. Disease review

Acharya Charaka has described the disease Kamala (jaundice) under the chapter of ‘Panduroga chikitsa upakrama’ [6]. Acharya mentioned different nidanas (causes), bheda (types), lakshanas (symptoms) and Chikitsa (treatment)for the disease Kamala. Kamala related to Balaka (infant) is due to ingestion of Dushtha Sthanya (vitiated breast milk) described in Charaka Samhita[11] and Madhava nidana [7] (Table 2).

In Asthatagni Hridaya sutrasthan, disease Kamala is formed due to increased rakt dhatu (blood plasma) [8] (Table 1).

There is a detailed explanation of nidana (causes), chikitsa (treatment), lakshana (symptoms) of kamala vyadhi (jaundice) in Yogaratnakara text. For the first time, there is an explanation of dronpushpi swarasa anjana (eye application) in kamala [9]. The above text also provided an explanation regarding stanya dushhti (vitiation of breast milk) by various doshas (bodily humors) and their symptoms with management, where the author described Kamala as a symptom of pittaja stanya dushti (breast milk vitiated by Pitta). Under the same, Acharya explained the use of Gaduchi, Shatavari, Patola patra, Nimba twak and Rakhcthandana processed with sharkara in pittaja stanya dushti to mother and child [9].

Acharya Kashyapa described the Lakshana of Kamala Vyadhi (symptoms of jaundice) related to balaka (infant) in Vedana Adhyaya, Sutrasthan of Kasypya Samhita. But the Nidana (causes), Samprapti (pathogenesis) and Chikitsa (treatment) are not been mentioned in Kashyapa Samhita.

2.3. Stany sthiti – a source for neonatal hyperbilirubinemia

Milk is defined as the essence of Rasa dhatu (plasma), which in turn depends on the diet and its assimilation in the mother. A navajata shishu (newborn) is dependent on to his mother for the food and nutrition. Therefore, breast milk if gets vitiated by the doshas (body humors), manifests various diseases as per the predominance of the doshas. The causes of vitillation of milk can be due to the defective dietetic intake in quality and quantity in the mother (Excessive use of snigda (unctuous), abhishyanda (food which increases kapha) and guru (heavy) substances like paayasyam or over intake of katu (spicy), amla (sour), lavana (salty) and kshara (alkali) substances) and also due to the defective eating habits and impaired digestion in mother [12]. Kashyapa describes number of grahamala to also vitiate milk: Shakuni making the milk acrid and bitter and Pootana bring sweet and acrid taste in milk.

2.4. Pittaja stanya dushti– an origin to neonatal hyperbilirubinemia

Milk vitiated by Pitta either gets discolored (Vivarmata) or acquires a disagreeable fetid smell (Durgandha) with more waste metabolites or decomposed, as though, the milk is kept at room temperature for long time. “Acharya Charakata” stated- The diseases of children and that of adults are quite similar but the difference is only in the dosha dushya, which are in smaller quantity than that of the adult [12]. Hence, the disease will be reviewed as per Charaka’s description for adults in consideration for the neonates. In Ayurveda Pandu roga (anemia) and Kamala are described together [12]. Stage 1- Pandu (5 types of Pandu according to dosha pradhamaata (predominance of body humors) explained by Charakacharya).

In above mentioned conditions, or even without Pandu roga (anemia) when one indulges in Ateeva Pittavardhaka Ahara (food that causes Pitta vitiation), kamala (jaundice) ensues in stage 2 (a) Vataja pandu with added Pitta produces Halemaka; (b) Pittaja pandu with added Pitta produces Kamala and (c) Kaphaja Pandu with added Pitta produces Kumbhakamala.

---

Table 1
Showing dhatu parinama (tissue formation).

<table>
<thead>
<tr>
<th>Fuel</th>
<th>Dhrtwagmi</th>
<th>Prasada paka (essence)</th>
<th>Kittapaka (waste)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poshaka Rasa</td>
<td>Rasagni</td>
<td>Poshya Rasa</td>
<td>Rakta</td>
</tr>
<tr>
<td>Poshaka Rakta</td>
<td>Raktagni</td>
<td>Poshya Rakta</td>
<td>Mansa</td>
</tr>
</tbody>
</table>
3. Etiopathogenesis

Severe depletion of ‘Ojaso gunaah’ (essence) in body (like bala, varna, sneha etc.) leads to alpa rakta (anemia), alpa meda (loss of fat) and nissara (less essence) which in turn causes shithila indriya (weakness of the sense organs) [12]. Such child attains nissara and Pittakara Ahara-Vihara Pitta (muscle tissues) and 2. Group 2: Vitiation of 3. Group 3: Various hemorrhagic conditions leads to neonatal hyperbilirubinemia (discoloration) of skin like varna (weakness of the sense organs) [12]. Such child attains nissara and Pitta (muscle tissues) and 2. Group 2: Vitiation of 3. Group 3: Various hemorrhagic conditions leads to neonatal hyperbilirubinemia (discoloration) of skin like varna (weakness of the sense organs) [12]. Such child attains nissara and Pitta (muscle tissues) and

3.1. Koshthashrita kamala and Shakhshashrita kamala in relation to neonatal hyperbilirubinemia

Koshthashrita kamala: Pandurogi (anemic) or by ingestion of Pittakara Ahara-Vihara leads to Pittaprapakpa (excessive vitiation of Pitta). That Vitiated Pitta produces Vidaha (burning of Mamsa (muscle tissues) and Rakta (blood). Vitiated Pitta gets accumulated in liver and then spread all over the body causing Bahupitta Kamala (hyperbilirubin) or koshashtra Kamala [12]. In Koshtashtra Kamala, the excessive Pitta pradhan dhatus (Pitta predominant body humors and tissues) leads to gauravam (heaviness) and prakopa (excessive vitiation of Pitta) which more bilirubin is found in blood due to excessive destruction of RBC and is not excreted adequately by liver resulting in hyperbilirubinemia. In modern science, it can be compared with hyperbilirubinemia due to prehepatic jaundice or Haemolytic jaundice in newborn can be considered. In conditions where there is depletion of ojo guna (essence of body) occurs due to 3 group of causes:

1. Group 1: Depression of RBC synthesis (dysshaemopoietic) due to excess intake of lavana, katu and Kashaya rasa (astringent taste)-can be considered in terms of dushta stanya paana (intake of vitiated milk) [13] and Pica/mud eating-cannot be considered in neonatal hyperbilirubinemia.

2. Group 2: Vitiation of Pitta pradhana doshas and dhatu (Pitta predominant body humors and tissues) leads to gauravam (heaviness) and shithilata (loosening) of that particular dhatu which further leads to depletion of ojo guna. Various metabolic and hemolytic causes are Maireyamadya sevana (alcohol intake) (cannot be considered in neonatal hyperbilirubinemia) and Daurgandhya stanya sevana (Pittajana dushti).

3. Group 3: Various hemorrhagic conditions leads to rakta kshaya (depletion of blood)- cephalhematoma and sub-galeal hemorrhage in newborn can be considered. In conditions where there is depletion of ojo guna, if one indulges in Pittavardhika ahara, Vayu through the ten dhanaanees (blood vessels) brings and lodges the pitta in twak (skin), mamsa (muscle) and rakta. This pitta burns these dhatus and produces different kamala (based on dosha). Thus kamala can occur in two ways: (a) Excessive Pitta in Pandu- Koshtashrita or Shakhshashrita kamala. (b) Without Pandu roga also as Pittobhana (due to increased Pitta) Therefore, Kamala is Pitta dosha pradhana vyadh.

Please cite this article as: Rathore S et al., A critical review on neonatal hyperbilirubinemia-an ayurvedic perspective, J Ayurveda Integr Med, https://doi.org/10.1016/j.jaim.2018.08.006
Vegadharana (suppressing natural urges) etc. In obstructive jaundice, same mechanism can be observed in which the bile ducts are obstructed by gall stone or other causes and bile is accumulated in liver, resulting in elevation of blood bilirubin level responsible for yellowness of eye, skin, mucous membrane and stool become clay colored due to lack of bile in the intestine. In Hepatocellular jaundice, when there is complete obstruction of all the bile canaliculi due to their compression by edematous hepatocytes, jaundice is produced just like shakhashrita Kamala. When there is incomplete obstruction or when all the bile canaliculi are not obstructed then it is produced like that of koshthashrita Kamala.

3.2. Physiology in relation to neonatal hyperbilirubinemia

Raktotpatti (Erythropoesis): Rakta Dhatu (blood) is stated to be formed from Rasa Dhatu (plasma) through Dhatu Purinama karma (formation of body tissues). The Dhatuparinama, regular nourishment of the Dhatus of the body, comprise of two pakas [18]. (a) Prasada paka (nourishment)-Leads to production of 7 type of Prasada dhatu. (b) Kitta paka (waste)-Leads to production of kitta or mala of respective dhatu. Three factors participating in parinama of Rasa dhatu to Rakta dhatu, are Rasagani (fire of plasma), Raktagati (fire of the blood) and Ranjaka pitta. Rasagani (fire of plasma) act on Ahara Rasas and leads to formation of Prasad (Rasa and Rakta dhatu respectively) and Kitta bhoga, i.e. Pitta (Ranjaka Pitta).

During the process of Dhatuparina (transformation of the tissues), Dhatwagni (factors responsible for tissue transformation) leads to the formation of Raktajeevaparmanu (elements of blood). Raktastra strotas and Raktdhara kala both helps in formation of Rakta Dhatu and Yakritra is the moola (site of origin). But ranjana karma (providing color) of Rakta is done by Ranjana pitta situated in Yakrita (liver), Pitha (spleen), and Amashaya (stomach) produced from poshaka Rakta as a malarupa [20]. As kamala (jaundice) is a Rakta pradoshaja vyadhi (disorder due to vitiation of blood) having Raktagati strotvakara (vitaion in blood channels), so Yakritra (liver) and Pleeha (spleen) are also important factors involved in the samprapti (pathogenesis) of the disease Kamala.

Relation between Dosa (body humors) and Yakrut (liver): Pitta dosha is formed in Yakritra. It is the primary station of Ranjaka Pitta. This is the factor involved for the formation of Rakta dhatu (blood) from Rasa dhatu (plasma). Therefore, here the hemolysis of the RBC’s can be considered giving rise to the production of bilirubin.

Relation between Dushya (body tissues) and Yakrut (liver): Yakritra is formed from Rakta Dhatu and it is the moola sthana of Raktavahastrotas [21]. Formation of Rakta Dhatu takes place in Yakritra. According to the consideration of Acharya Sushruta and Vagbhata, the Yakritra is derived from the accha (pure) portion of the fetal blood [22]. Hence, the structure is soft, well organized and secretary in nature. It secretes Pachaka pitta that is stored in the Pittashaya (duodenum) or the gall bladder. The concentration of Pitta is very important. If the concentration alters, it leads to lot of diseases arising out of Agniavishaya (vitiating of digestive fire). Here, Yakritra can be compared with the organ Liver of the contemporary medicine.

Pitta (Malarupa): Tejas (fire element) is present in the form of Agni, which is present in our body in the form of Pitta [23]. Manifestation of yellow color to body is due to Pitta vrudhi which is due to tejo mahabhuta (fire element). Kamala is a disease which is due to Pitta vrudhi. Hence, vitiation of pitta and agniavishayamaya (impairment of digestive fire) affect each other and vice versa. Moolashana of Raktaavahastrrotas Yakritra and Pleeha. Therefore, this can be understood that atpatti of malarupittra (bile) takes place in Yakritra with the help of Raktagati. Because of samana guna (same properties) of Pitta and Rakta, Pitta are main along with Rakta in ashrayashrayi bhava (association) [24]. When agniavishaya (impairment in the digestive fire) happens in the body, then it produces malarupi pitta in larger quantity which is stored in yakrita and circulated all over the body and gives vikruta varna (discoloration) to the body.

4. Clinical features of Kamala

4.1. Pathogenesis of neonatal hyperbilirubinemia in the view of Ayurveda

Due to the Nidana sevana (causative factors), Pitta pradhana tridoshas (Pitta predominant body humor) gets aggravated, which in turn vitiates the Rakta dhatu (due to ashraya ashrayi sambandha). After Rakta dhatu (blood) getting vitiated, the moola of Rakta dhatu i.e. Yukrut and Pleeha, also gets vitiated and therefore, the Rakta dhatu kshaya (decreased blood) takes place (both in quality and quantity-quality is maintained by the yukrut by doing the ranjana karma which can be compared with the conjugation of the unconjugated bilirubin -> gets hampered). Pleeha maintains the quantity of rakta dhatu which can be compared with the early lysis of the RBC’s. This leads to the shithilata (impairment) of the Rakta and Mansa dhatu and therefore, the aggravated pitta due to atipravritti (excessive production) and vimargagamanam (movement in opposite direction), takes shrama samshraya (location) at tvak (skin), rakta (blood) and mansa dhatu (muscle tissues) and manifest as Neonatal hyperbilirubinemia (Fig 1).

Samprapti Ghataka (factors responsible for pathogenesis) [25].

1. Doshas: Pitta
2. Dushyas: Rakta, Mansa
3. Adhisthanas: Kostha (Maharotasa — Yakrit), Shakra (Raktadi and tvach)
4. Srotas: Rasavaha, Raktaavaha, Annavaha, Pureeshavaha
5. Srotodudhi: Atipravritti (excessive production), Sanga (obstruction), Vimargagamanam (moves in opposite direction) - (in physiological neonatal hyperbilirubinemia, atipravritti and vimargagamanam can be considered whereas in pathologic neonatal hyperbilirubinemia, sanga, atipravritti and vimargagamanam can be undertaken).

5. Prognosis

While describing the disease kamala of the Acharyas have mentioned its Sadyija-Asadhyaya, whereas almost all the Acharyas has mentioned that negligence or improper management of Kamala leads to complications which can be taken as the conditions like kernicterus, brain encephalopathy, chorio-athetoid cerebral palsy in the contemporary science. Ayurvedic classics mentioned the following as the Asadhyaja lakshanas (untreatable symptoms) of kamala, indicating bad prognosis like Krishnapeta mutra and shakrit (dark yellow colored urine and stool), Atishotha (edema), Raktakshita (redness of eye), Raktmurta (hematuria), Daha (burning sensation), Aruchi (loss of taste), Trishna (thirst/dehydration), Anaha (indigestion), Tandra (tiredness), Nashagati (loss of appetite), Nashita Sanyya (unconsciousness) etc.

So, Kumbha kamala can be compared with that of the Kernicterus explained in modern science as the complication of neonatal hyperbilirubinemia.

6. Treatment

There is no direct reference of Navajata Kamala chikitsa (neonatal hyperbilirubinemia) in Ayurveda, but, as the nidana for navajata kamala is considered to be pittaja sthanyu dushhti, which is consumed by the baby, pittaja sthanyu dushi chikitsa is taken as the line of treatment. Generally correction of vitiated milk brings forth normalcy in the child in mild cases. In severe cases, the child can be
administered medicine depending on the vitiated *dosa*. *Sushruta* in *chikitsa sthana* advocates induction of vomiting to the mother irrespective of the vitiated *dosa* in milk. *Charaka* though advocates induction of both vomiting and purging in *chikitsa sthana* 30th chapter, advises one or more of the four means of commingling i.e. of *Vamana* (vomiting therapy), *Virechana* (purgation therapy), *Asthapana* and *Anuvasana* (enema therapy), depending on the *doshas* vitiated [12]. Thus, the principles of treatment of vitiated milk disorder can be classified as follows:

6.1. In mother

General measures taken are (a) Extirpation of the vitiated humour by induction of vomiting irrespective of the *Dosha* with oral use of the decoction of *Nimba* with honey and *Pippali* as advised by *Sushruta* [12], (b) *Pathya Bhojana* [26], (c) Internal medication to the mother [27] consists of combination of different drugs selected from among the groups of *Vamana* (vomiting therapy), *Virechana* (purgation therapy), *Asthapana* and *Anuvasana* (enema therapy), depending on the *doshas* vitiated [12]. Thus, the principles of treatment of vitiated milk disorder can be classified as follows:

![Pathogenesis of neonatal hyperbilirubinemia through Ayurveda](https://doi.org/10.1016/j.jaim.2018.08.006)

Please cite this article as: Rathore S et al., A critical review on neonatal hyperbilirubinemia-an ayurvedic perspective, J Ayurveda Integr Med, https://doi.org/10.1016/j.jaim.2018.08.006
of Daru, Musta and Patha to which Saindhava is added. Slimy milk can be made good by oral use of Takarishita or of Abhaya, Vacha, Musta, Shunthi and Patha. A poultice made of Vidari, Bilva and yashthi is applied to the mammary glands in both the conditions and milk is removed [12]. Heavy milk can be made light by oral use of either the decoction of Trayaman, Anamrita, Nimba, Patola and Triphala or the paste of Pippalimoola, Chavya, Chitraka and Shunthi. A poultice made by Bala, Shunthi and Moorva or of Prishnaparni and Payasya applied to the breasts and washed after it becomes dry makes the milk light [12].

6.2. In child

In mild cases, no treatment to the child is required and hence Sushruta and Agnivesha do not advocate treatment for the child normally when vitiated milk is sucked. The ailment becomes corrected with the treatment of the morderalone. However, Vagbhata gives description of treatment to the child and this should be applied when humors get vitiated more. When milk becomes vitiated with Vayu, the child is given the powder or ghee prepared of Rasna, Ajamoda, Sarala and Devadara to which sugar may be added [28]. In Pittaja milk disorder, the decoction of Amrita, Abheeru, Patola, Nimba, Chandana and Sariva is given to both, the mother and the child [29]. After applying the paste of Raathu-pushpa to the nipple and areola, the child is made to suck it without being washed to bring forth an easy emetic effect in cases of vitiation of milk by Kaphadosha [12].

6.2.1. Anulomana and mrduvirechana

In Kamala vyadhhi, Virechana is the main treatment. In pittaja vimàra also, the main principle of treatment is Virechana. Acharyas mentioned anulomana (correcting the direction of Vata) and Mrudu virechaka drugs for virechana karma in Kamala and Pittaja Vikaara. In Balaka, Virechana like Panchakarma is contra-indicated but Mrduvirechana (mild purgative therapy) and Anulomana karma are mentioned [30]. The following formulations mentioned under the disease Kamala (Jaundice) as mentioned in classics can be tried in newborns in an appropriate and palatable form (Table 3).

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Showing Shammanushadhis in Kamala vyadhhi.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single drugs</td>
<td>Amrita Swarasa</td>
</tr>
<tr>
<td></td>
<td>Bhunimbamalaki Swarasa</td>
</tr>
<tr>
<td></td>
<td>Daruharidra Swarasa</td>
</tr>
<tr>
<td></td>
<td>Nimbapatra Swarasa</td>
</tr>
<tr>
<td>Compound drugs:</td>
<td></td>
</tr>
<tr>
<td>A. Churna</td>
<td>Navayasa Churna</td>
</tr>
<tr>
<td></td>
<td>Bhunimbadi churna</td>
</tr>
<tr>
<td></td>
<td>Jeeruvantyadi Churna</td>
</tr>
<tr>
<td>B. Kwatha</td>
<td>Phalatrikadi kwatha</td>
</tr>
<tr>
<td></td>
<td>Vasadi kwatha</td>
</tr>
<tr>
<td>C. Avaleha</td>
<td>Darvyadi lehya</td>
</tr>
<tr>
<td></td>
<td>Triphaladi Avalehya</td>
</tr>
<tr>
<td></td>
<td>Drakshadi lehya</td>
</tr>
<tr>
<td></td>
<td>Vidanga Lehya</td>
</tr>
<tr>
<td></td>
<td>Swarnamaksheka</td>
</tr>
<tr>
<td></td>
<td>Vidangadi Loha</td>
</tr>
<tr>
<td></td>
<td>Punarnava Mandura</td>
</tr>
<tr>
<td></td>
<td>Mandura bhasma</td>
</tr>
<tr>
<td>D. Rasasoushadhi</td>
<td>Dhati Loha</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>E. Ghritas</td>
<td>Kalyanaka Ghrita</td>
</tr>
<tr>
<td></td>
<td>Mahakutki Ghrita</td>
</tr>
<tr>
<td></td>
<td>Panchgavya Ghrita</td>
</tr>
<tr>
<td></td>
<td>Pippalyadi Ghrita</td>
</tr>
<tr>
<td></td>
<td>Trayushanadi Ghrita</td>
</tr>
</tbody>
</table>

7. Role of MRP 2 molecule and mode of action of drugs

The pharmaceutical action of the ayurvedicdrugs can be understood in two ways i.e. increasing the uptake of the unconjugated bilirubin for its conjugation by the hepatocytes, enhancing the transportation of the conjugated bilirubin to the intestine and then the excretion of the conjugated bilirubin [31]. The increase in the uptake, conjugation and its transport can be understood by the role of MRP 2 molecule. The multi-drug resistance protein MRP2 is an ATP-binding cassette transporter playing an important role in detoxification by transporting a wide range of compounds, especially conjugates of lipophilic substances with glutathione, glucuronate and sulfate, which are collectively known as phase II products of biotransformation. In addition, MRP2 can also transport unchanged compounds in cotransport with glutathione, and thus can modulate the pharmacokinetics of many drugs. For the excretion of the conjugated bilirubin, the rechaka (purgative) property of the drug helps to prevent the rise of serum bilirubin level. The above mentioned drugs for the treatment of Kamala act upon the uptake of unconjugated bilirubin by the hepatocytes, stimulating the MRP 2 protein molecule for the quick transport of the conjugated bilirubin for its excretion by the rechaka property. The drugs by their Pittahara and rasayana (rejuvenative) property help in reducing the formation of malarupi Pitta (bile) and also in the regeneration of the yakrut (hepatocytes) for its uptake. The antioxidant, antimicrobial and immunomodulatory property of the drugs helps in scavenging the free radicular stress, thereby, prevent the rise in the level of bilirubin.

8. Conclusion

The concept of neonatal hyperbilirubinemia in Ayurveda can be understood in the context of Pittata sthaya dushti along with the physiological variations in the newborns leading to the raised level of unconjugated bilirubin. Therefore, the patho-physiology should be known by a pediatrician in Ayurveda based on the involvement of dosha, dhatu, mala and srotas. The standard treatment principle mentioned in the contemporary science as phototherapy has its own side-effects [32]. Therefore, Ayurvedic pediatricians should bring forth formulations to prevent the rise of bilirubin to an extent to cause complications like kernicterus etc.

References


